Angiographic Correlate of Post-Reperfusion Abnormal Q Waves

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SUMMARY

To determine whether post-reperfusion acute Q waves are due to irreversible myocardial necrosis, we studied the relationship between abnormal Q waves and left ventricular regional wall motion in 74 patients who had coronary thrombolysis following acute myocardial infarction. In 48 cases, acute pathologic Q waves appeared on the ECG-recordings after coronary reperfusion (group A), whereas in 26 patients the QRS complex had no or only minimal changes (group B). A control group consisted of 27 patients with unsuccessful thrombolysis. Quantitative left ventricular angiography was performed after coronary thrombolysis and repeated before discharge from the hospital. Regional wall motion of the infarcted area was determined by a system of 48 radii traced from the centroid of the end-diastolic and end-systolic silhouettes.

Myocardial akinesis was significantly more extensive in group A than in group B ($18 \pm 9\%$ vs. $10 \pm 6\%$, $p < 0.02$). Regional wall motion of the infarcted area improved in both groups at predischARGE study (mean radial shortening $+5 \pm 6\%$ in group A and $+4 \pm 7\%$ in group B). Moreover, regional wall motion was significantly better in group A with respect to the control group (angio score of hypo-akinesis 166 ± 124 vs. 412 ± 174 , $p < 0.01$).

In conclusion, post-reperfusion abnormal Q waves 1) are associated with more extensive myocardial damage, 2) do not preclude late recovery of ischemic myocardium, and 3) do not necessarily indicate irreversible transmural necrosis.

Additional Indexing Words:

Coronary thrombolysis	Post-reperfusion Q waves	Electrocardiogram
Myocardial wall motion	Myocardial infarction	

THE abrupt occurrence of abnormal Q waves has been frequently described after coronary reperfusion.¹⁾⁻³⁾ However, the pathophysiologic correlate of this finding is still unresolved.

Although accelerated cellular destruction has been demonstrated follow-

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ing coronary thrombolysis,⁴⁾⁻⁶⁾ the equation of post-reperfusion Q waves with myocardial death lacks proof. Accordingly, prior pathologic studies have shown that chronic Q waves do not reliably estimate the extent of myocardial infarction.⁷⁾⁻¹⁰⁾

Moreover, even small infarcts have been associated with abnormal Q waves,^{7),9)} suggesting that a substantial amount of still viable myocardium may be present in the underlying ventricular wall.

It is important to assess if post-reperfusion Q waves reflect irreversible myocardial damage and/or represent a marker of late coronary thrombolysis. In addition, we must decide if extensive Q waves contraindicate surgical revascularization of the infarct related vessel.

In this study we sought to clarify the correlation between rapid post-reperfusion Q waves and angiographically documented myocardial damage. Furthermore, we determined whether the development of new Q waves indicates inefficient thrombolysis.

METHODS

Seventy-four patients with acute transmural myocardial infarction, in whom thrombolysis was performed to recanalise the obstructed coronary artery, were studied. They met the following criteria: a) acute prolonged chest pain lasting less than 3 hours; b) no history or echographic evidence of previous myocardial infarction; c) total occlusion of a subepicardial coronary vessel at early angiography; d) coronary reperfusion within 60 min from the start of intracoronary fibrinolytic therapy; e) sustained patency of the infarct-related artery at control pre-discharge angiography.

Not accepted for the study were 1) patients with left bundle branch block; 2) patients who had pathologic QS complexes on the admission ECG; 3) patients who showed "ischemia at a distance" (recognized as "nonspecific" ST-T abnormalities and/or mechanical dysfunction of the noninfarcted regions).

According to the post-thrombolysis ECG-findings, the study patients were subdivided as follows: group A consisted of 48 subjects who showed new significant Q waves and group B included 26 patients who had no or only minimal changes in the QRS pattern (small Q waves or reductions in R wave amplitude).

Diagnosis of new pathologic Q waves was based on a) Q-duration $>0.03''$, b) Q-amplitude >3 mm, c) presence of QS complex or Q/R ratio >1 in at least two ECG leads.

In addition, the ECG score based on the system of Wagner¹¹⁾ was cal-

culated. Because the ECG rapidly evolves after coronary thrombolysis, the ECG tracing analysis was performed from the 10th–15th day when the pattern was stable.

The control group consisted of 27 patients who did not achieve coronary patency or who had vessel reocclusion at control angiography (group C). There was no significant difference between groups with respect to age, sex, infarct location and time from the onset of symptoms.

Quantitative analysis of regional wall motion:

The analysis of regional wall motion was performed during left ventricular angiography. Opacification of the left ventricle was achieved with a power injection of 30–40 ml of nonionic contrast medium (Iopamiro, Bracco). The cinefilms were recorded in the right anterior oblique projection and exposed at a speed of 48 frames/sec by means of a 35 mm cinecamera (Arryflex-Arrytechno, München).

The angiograms were interpreted independently by 2 experienced observers. End-systolic and end-diastolic contours from the first fully opacified beat were manually traced on a paper sheet at about a three fold magnification. Post-extrasystolic beats were not considered. Ventricular volumes were obtained by the area-length method of Dodge.¹²⁾ Left ventricular ejection fraction was derived from the formula: end-diastolic volume minus end-systolic volume / end-diastolic volume. The length of the akinetic segment was expressed as a percent of end-diastolic perimeter according to the method of Feild.¹³⁾ Quantitative analysis of regional wall motion was performed by a system of 48 radii traced from the centroid of the end-systolic and end-diastolic contours, excluding the aortic valvular plane. After having superimposed the left ventricular silhouettes, systolic fractional shortening of each segment was measured. Hypo-akinesis was defined as a fractional shortening less than 25%. Outward systolic displacement was represented by a negative change.

The first two radii close to the aortic valve and the last three radii corresponding to the mitral plane were not considered because of their great variability.

Regional wall motion of the infarcted area was computed as the mean fractional shortening of radial segments in the abnormally contracting area. An angio index of myocardial systolic dysfunction was derived from the product of the mean shortening of the infarcted area and the number of involved hypo-akinetic segments.

Study protocol:

The following protocol was used:

- 1) left ventricular angiography immediately after the end of thrombolytic

- therapy (early study of regional wall motion),
- 2) clinical observation for 10–15 days to allow a complete restoration of left ventricular function,
 - 3) control left ventricular angiography before hospital discharge (late study of regional wall motion).

Three different analyses were carried out. First, angiographic extension of the infarcted area was compared between group A and group B. Second, the presence or absence of post-thrombolysis Q waves (groups A and B) was correlated with the change in systolic wall motion of the infarcted area (within-group analysis). Third, left ventricular wall motion was compared between patients with rapid post-reperfusion Q waves (group A) and patients with late “traditional” Q waves (group C).

Between-group comparisons were carried out with Student's t-test and within-group comparisons with a t-test for paired series.

RESULTS

Relationship between post-reperfusion Q waves and infarct size:

As reported in Fig. 1, a positive but poor correlation was found between post-reperfusion Q wave amplitude (group A) and the angio index of myocardial damage ($r=0.46$). The relation for the entire group was not significantly improved when we considered the ECG scoring system of Wagner ($r=0.51$) (Fig. 1) or the number of Q/R and QS complexes. Only in the subgroup with anterior Q wave infarction did the linear regression analysis demonstrate a fairly good correlation with the Wagner score ($r=0.68$).

Follow-up changes in left ventricular regional wall motion:

Post-reperfusion extension of the hypo-akinetic area was significantly higher in group A compared to group B ($p<0.02$). At follow-up angiography, however, the regional wall motion of the infarcted area was found to improve similarly in group A (mean radial shortening $+5\pm6\%$) and group B (mean radial shortening $+4\pm7\%$) (Fig. 2).

Comparison between “abrupt” Q waves and “late” Q waves:

Group A and control group C showed a similar amplitude and number of Q waves. The ECG scoring system of Wagner was also superimposable (Table I). However, the angio index of myocardial damage was significantly less in group A (166 ± 124) with respect to group C (412 ± 175). It appeared that the difference observed between the 2 groups was still present and significant even when the length of the akinetic segment or the mean shortening of the infarcted region was considered (Table II).

Fig. 1. Correlation between the total amplitude of acute post-thrombolysis Q waves and angio index of myocardial damage (upper panel). In the lower diagram the relationship between the ECG scoring system of Wagner and the angio index is reported.

DISCUSSION

In the past, the appearance of pathologic Q waves was considered the marker of irreversible myocardial damage.^{14),15)} The rational basis of this statement was established by Wilson,¹⁶⁾ who attributed the genesis of abnormal Q waves to the direct recording of the negative endoventricular potentials through "the electrical window" of a necrotic area.

More recently, several studies have shown that abnormal Q waves are a nonspecific finding.¹⁷⁾

Transient Q waves have been observed in patients with acute myocardial ischemia,¹⁷⁾⁻²⁰⁾ allergic shock,²¹⁾ pancreatitis²²⁾ or phosphorus poisoning.²³⁾ Moreover, chronic Q waves have been reported to disappear after coronary bypass surgery.^{24),25)} These observations imply that abnormal Q waves are not necessarily associated with tissue death and may be consistent with a significant amount of viable myocardium.

Fig. 2. Regional wall motion of the infarcted area at basal and control angiography. Note that the fractional shortening of the infarcted region improved in both thrombolysis groups, regardless of whether acute Q waves were present (group A) or absent (group B).

Table I. Electrocardiographic Data of Patients with Post-Thrombolysis Acute Q Waves (Group A) and "Traditional" Late Q Waves (Group C)

	Group A n=48	Group C n=27	
Number of new Q waves	2.2±1	2.5±9	p=ns
Total amplitude of new Q waves (mm)	13.6±5.3	15±6.6	p=ns
Wagner's score	6.8±3	7.3±3.3	p=ns

Table II. Angiographic Parameters of Myocardial Damage in Thrombolysis Q-Wave Patients (Group A) and Control Population (Group C)

	Group A n=48	Group C n=27	
Length of akinetic segment (cm)	5.8±2.7	11.6±3.3	p<0.01
Akinesis (%)	18±9	27±10	p<0.01
Mean shortening of the infarcted area	7.3±4.3	4.1±2.8	p<0.01
Angio index of myocardial dysfunction	166±124	412±175	p<0.001

However, the precise causes and the events which may be involved in the development of transient Q waves remain obscure.

Hypothetically, acute ischemia and various noncardiac diseases such as

electrolyte disturbances, anaphylactic shock etc., may determine a loss of transmembrane ionic homeostasis.²⁶⁾ As a result, damaged myocytes lose their ability to generate electrical forces. In these cases it is likely that the transient Q waves reflect electrical silence without biologic death.

In other circumstances, such as ischemia-induced right precordial Q waves, the mechanism seems to be a conduction delay in the activation of the septal wall ("septal focal block"²⁷⁾).

Significance of post-reperfusion Q waves:

Although there is a close temporal relationship between post-thrombolysis Q waves and post-reperfusion myocardial damage, the electrical phenomenon does not necessarily reflect a completed infarct.

In our experience, the new development of pathologic Q waves was associated with a higher percentage of abnormally contracting segments compared to the normal-QRS thrombolysis group. However, when the amplitude or extension of Q waves was correlated with the angiographically determined infarct-size, the results were not very encouraging. The correlation was so low that the clinical utility of the electrocardiogram for estimating the extent of the myocardial necrosis in an individual patient was quite limited.

As a consequence, the equation of myocardial infarction and Q waves is not always justified. In addition, we found that post-thrombolysis Q waves do not exclude a functional recovery of the infarcted area and that the regional wall motion improved to a similar extent in the patients with or without abnormal Q waves.

The possible explanations of these observations are uncertain. Although either "electrical myocardial silence" without actual biologic death²⁶⁾ or intraventricular conduction disturbances²⁷⁾ has been advocated as a possible mechanism of non-necrotic Q waves, these phenomena do not explain the occurrence of post-reperfusion Q waves. The loss of electrical forces is a very unstable condition which rapidly evolved towards tissue death or else towards metabolic and electrical recovery. In contrast, the post-reperfusion Q waves appear to be persistent.

On the other hand, an intraventricular conduction delay may be responsible for the appearance of right precordial Q waves, but does not account for all such cases.

It is important to stress that the degree of extension of akinesis at follow-up angiography showed a 50% difference in favor of the post-thrombolysis Q-wave patients compared to the control group. Nevertheless, there was no difference in the Wagner ECG score or in the total amplitude of Q waves between the 2 groups.

This implies that a similar electrocardiographic pattern may be consis-

tent with different amounts of damaged tissue. Moreover, our cases provide documentation that post-thrombolysis Q waves are associated with a lower degree of myocardial necrosis compared to that associated with late "traditional" Q waves.

To understand this fact we must recognize that an abnormal Q wave may appear when only the subendocardial layer is infarcted. Accordingly, Maxwell et al²⁸⁾ have shown that similar QS or QR patterns could be recorded regardless of whether the infarct was transmural or tended to involve primarily the subendocardial region.

It would be reasonable to assume that myocardial reperfusion may prevent necrosis of the superficial layer, but not the inner myocardium.²⁹⁾ In such cases, significant Q waves, indistinguishable from those of transmural infarction, can be recorded on the surface electrocardiogram.

Conclusions:

In conclusion, coronary thrombolysis is associated with a substantial improvement in the regional wall motion of the infarcted area. Although acute post-thrombolysis Q waves are observed more frequently when the myocardial damage is greater, the functional restoration of the myocardial wall does not influence the appearance of new Q waves.

Thus, the post-reperfusion Q waves a) do not necessarily indicate a completed infarction, b) are not a marker of late thrombolysis, and c) do not exclude a surgical attempt at myocardial revascularization.

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